***Helicobacter pylori* (peptic ulcer disease)**

**Signs and symptoms for the disease it produces.**

* Abdominal pain
  + Often improves following meals or taking antacids
* Heartburn
* Midepigastric tenderness
* This pt worked under stressful conditions

**The source of infectious organism.**

* *Helicobacter pylori*

**The manner of exposure, route of infection, tissues that they reside and, where appropriate, transmission to others.**

* Worldwide
  + In developing countries, more than 80% of people are affected (30% in US)
* Humans and monkeys are only known habitats
* Fecally contaminated water causes more frequent colonization in developing countries
* **Increased age** and **poor socioeconomic conditions** are the most important risk factors for overgrowth and chronic infection
* Exacerbating factors: excess acid, smoking, stress, spicy foods

**The pathology and the manner by which the particular disease develops and/or is induced, including damage caused by the pathogen and damage caused by the immune system’s response to the pathogen.**

* Predominant features:
  + Only found on gastric epithelium
  + **Does not invade** cells
  + **Elicits robust inflammation in stomach (chronic gastritis)** and immune response
  + Is rarely spontaneously cleared
* **A cloud of ammonia (w/ buffering protection) produced by urease activity (a major virulence factor)** protects the organisms from the gastric acid
* Flagella and mucinase allow them to pass through the mucus layer
* Bacteria are anchored at intracellular junction of enteric cells to the epithelial cells by attachment pedestals
* **Other major virulence factors:**
  + **VacA – vacuolating cytotoxin**
  + ***Cag* pathogenicity island (*cag* PaI) genes**
    - CagA protein is an identifiable marker of inflammation
* Chronic gastritis is characterized by a **mononuclear inflammatory cell infiltration associated with neutrophilic infiltration into the lamina propria**

**Methods of identification and placement into a particular biological subset.**

* Histologic exam: based on endoscopic biopsy specimens
* Culture: based on endoscopic biopsy specimens
* **Fecal antigen test**
* **13C urea-breath test**
* **Serology to detect specific IgG** (response is primarily IgG)

**Gram negative curved rod.** Motile, makes **urease**. Growth needs selective media with nutrient supplements, **microaerophilic** conditions, and an incubation temp of 37 C for up to 10 days.

**Factors leading to enhanced resistance or susceptibility (e.g., recipients of vaccines, residence in geographic areas, types of work, immunodeficiency, alcoholism, age, violence/abuse, religious beliefs, etc.).**

* Living in a developing country
* Getting older
* Having shitty socioeconomic conditions

**Other organisms in the differential diagnosis and how to discriminate among potential causative agents.**

* Appendicitis
* Cholecystitis
* Cholelithiasis
* Crohn’s disease
* Esophagitis
* Gastroenteritis
* Gastroesophageal reflux

Lower-right quadrant pain would suggest appendicitis, upper-right quadrant pain would suggest cholecystitis or cholelithiasis. No diarrhea or emesis = prob not Crohn’s. If it was esophagitis or acid reflux, chest pain would be a predominant symptom.

**Prevention, treatment and vaccine design (live vs. dead).**

Appropriate antibiotic regimens can eradicate it, which permanently cures ulcers in many cases. Most successful regimens use 2 antimicrobials + PPI for 2-4 weeks.

* **Metronidazole or tetracycline** or **amoxicillin** in addition to **clarithromycin and omeprazole** (a PPI)

No known preventative measures.

FYI: In untreated cases **duodenal ulcers** and gastric B-cell (**mucosa-associated lymphoid tissue [MALT]**) **lymphoma** are likely complications